REVIEW

Genital allergy

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Genital allergy should be considered as a possible diagnosis in all patients with genital soreness or irritation for which no infection or dermatosis can be identified and in whom symptoms remain unchanged or worsen with treatment. Type I and IV hypersensitivity reactions are most commonly encountered and can be assessed by performing skin prick testing/radioallergosorbent test (RAST) or patch testing, respectively. Type IV reactions (contact dermatitis) may sometimes prove difficult to distinguish clinically from an irritant dermatitis. This clinical review attempts to summarise key features of genital allergy for the practising clinician.

Genital soreness and irritation are common symptoms and in the majority of cases an infective or common dermatological cause can be identified. Occasionally the diagnosis proves a little more elusive and trials of antifungals and topical steroids are unsuccessful. It is in such patients that the possibility of genital ''allergy'' should be considered. There are four recognised types of immunological hypersensitivity reaction and types I, III, and IV have been reported to affect the genitalia or genital tract (table 1). When considering genital skin reactions it is important to distinguish between irritant problems that result from a direct effect of the substance concerned with the genital epithelium in the absence of an allergic mechanism and true contact dermatitis (that is, type IV hypersensitivity). Irritants may cause more intense reactions on the vulval epithelium than on non-genital skin, probably as a result of a higher transepidermal water loss, capacitance, and blood flow in the vulva.[1] Genital hypersensitivity reactions may be subdivided into those that are related to sexual ''activity'' (for example, kissing, foreplay, coitus) and those that may occur in the absence of sexual contact.

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SEXUALLY RELATED HYPERSENSITIVITY

Seminal fluid

In 1958, Specken reported the case of a 65 year old woman who suffered post-coital generalised urticaria at times accompanied by brochospasm. This was the first description of hypersensitivity to semen and over subsequent years a number of cases and series of cases have appeared in the medical literature. Symptoms may occur with first exposure to seminal fluid or after years of ''uneventful'' sexual intercourse and range from purely local to generalised systemic reactions.[2 3] Local responses consist of genital swelling, burning, irritation, or soreness which may occur during or soon after intercourse, usually becoming maximal at 24 hours and lasting 2–3 days.[4 5] Semen contact with non-genital skin may also give rise to localised itching and urticaria.[6 7] Generalised reactions associated with semen allergy include angioedema of the lips and eyelids,[6 8] laryngeal oedema,[9] bronchospasm,[10] and anaphylaxis[3 4 7] but, to date, death has not been reported. Semen allergy mainly affects younger women although postmenopausal cases are documented.[11 12] An increasing intensity of reaction with subsequent episodes of coitus is a common feature. Levine *et al* described a married woman with a 15 year history of hay fever who initially presented with swollen eyes, nasal congestion, and sneezing an hour after coitus. Ten days later she developed similar symptoms together with diffuse urticaria and a sensation of throat swelling 5 minutes post-ejaculation. During the next year her symptoms were prevented by using a condom or by coitus interruptus. On four occasions these precautions failed and symptoms developed.

Most affected women have a personal or family history of atopy,[3 8 11] although this is not always the case[2 8] and familial ''allergic seminal vulvovaginitis'' has been described affecting a mother and three daughters.[4]

The specific allergen(s) within semen responsible for triggering type I hypersensitivity is still unknown. Mumford *et al* described a woman with post-coital wheezing and dyspnoea who, for 3 months before these symptoms, had complained of perineal irritation.[12] Seminal plasma separated from sperm produced a positive intradermal skin test but a negative patch test. Both tests were negative with sperm only. Further analysis of the seminal plasma suggested that the sensitising agent had a molecular weight of between 14 000 and 30 000 daltons. Other studies have confirmed that the potential allergens are glycoproteins of molecular weight between 12 000 and 75 000,[9 13–15] and are probably derived from the prostate or seminal vesicles since vasectomy fails to prevent symptoms.[3 9]

A number of studies have found an association between the onset of seminal fluid allergy and genital tract ''procedures'' such as tubal ligation, hysterectomy, intrauterine contraceptive device insertion, and pregnancy.[1 3 6–8] It has been suggested that these events may in some way disrupt normal immunomodulation in the female genital tract,[16 17] although the precise mechanism by which this may occur has not been elucidated.

Hypersensitivity reactions to seminal fluid other than type I are less common. Type III

Table 1 Summary of the four types of hypersensitivity reaction

Type I Immediate hypersensitivity
Dependent on the specific triggering of IgE sensitised mast cells by antigen—for example, asthma, hay fever, urticaria, anaphylaxis
Type II
Antibody is directed against antigens on specific host cells and tissues—for example, graft rejection, autoimmuine haemolytic anaemia, myasthenia gravis
Type III
Antigen-antibody complexes are deposited in tissues—for example, rheumatoid arthritis, systemic lupus erythematosus, serum sickness, infective endocarditis, malaria
Type IV Delayed hypersensitivity
Antigen sensitisred T cells release cytokines following secondary contact with the same antigen—for example, contact dermatitis, tuberculosis, leprosy

(immune complex) hypersensitivity to seminal fluid has been reported in a young woman who developed nasal congestion and urticaria 8 hours after intercourse on her honeymoon.[18] She subsequently developed migratory arthralgia, periorbital oedema, dyspnoea secondary to a restrictive ventilatory defect, and a haemorrhagic proctitis. Investigations showed the presence of circulating immune complexes in the serum and evidence of complement activation.

There are no reports of pure delayed type hypersensitivity (DTH) reactions (type IV hypersensitivity) to seminal fluid although DTH reactions involving other factors may accompany type I hypersensitivity to seminal fluid.[11] An experimental model of contact sensitivity for the murine oral mucosa does at least provide some theoretical basis for DTH reactions affecting the genital tract mucosa.[19]

Spermicides
Contact dermatitis to spermicidal preparations is an uncommon but well recognised condition, possibly more commonly affecting men.[20] The sensitising agent may be one of the active compounds (for example, benzocaine, monophenoxypolyethoxy derivatives, hexyl resorcinol, chloramine, quinine, or an associated fragrance).[20–22] Nonoxynol-9 may cause genital soreness and irritation secondary to the compound's irritant properties[23] or as a result of contact dermatitis.[24]

Latex
Both type I and type IV hypersensitivity reactions have been reported to rubber products, including condoms. Commonly reported presentations include contact dermatitis, contact urticaria and, more rarely, anaphylaxis.[25 26] As with most natural allergens, the allergenic fraction of natural rubber latex varies in amount (as a result of factors such as climate, season, etc) and in polypeptide content. It is of interest to note that latex allergy may be associated with fruit allergy, in particular avocado, banana, kiwi fruit, melon, peach and less commonly fig, plum, chestnut, peanut, potato, papaya, and tomato.[27] Other potential allergens used during condom manufacture include carbamates and thiurams, although the latter tend not to be used nowadays.[28] ''Hypoallergenic'' condoms may contain lower amounts of additives but are not totally free of latex proteins and therefore should be used with caution in patients with true rubber latex sensitivity.[29] Individuals with latex sensitivity should be advised to use condoms made from synthetic materials, such as polyurethane. There have been no published reports to date of hypersensitivity reactions to the recently developed male polyurethane condom.

KY jelly
Contact dermatitis has been reported following the use of KY jelly and is the result of propylene glycol sensitivity.[30] Propylene glycol is widely used as a vehicle for cosmetics, body lotions, antiperspirants, and topical medicines and should be considered as a possible sensitising agent in patients with genital dermatitis without an obvious cause.

Oral medications
Ingested antigens may pass into seminal fluid and rarely produce a hypersensitivity reaction in the sexual partner. Haddad reported the case of a woman allergic to walnuts who developed an anaphylactic reaction after intercourse with her husband.[31] He had eaten walnuts before coitus and walnut protein was subsequently detected in his seminal fluid. Post-coital hypersensivity reactions have also been described in association with penicillin,[32] vinblastine,[33] and thioridazine ingestion.[34]

Topical preparations
Fisher reported the case of a young woman who repeatedly developed an eczematous eruption on her face, neck, and occasionally arms after sexual intercourse with her boyfriend.[35] Patch tests to commonly encountered allergens, including cosmetics, were negative. Further investigation revealed that her boyfriend used 5% benzoyl peroxide for facial acne. Subsequent patch testing showed her to be sensitive to this preparation and her eczema subsided after her partner changed to a topical antibiotic cream. A similar case of consort dermatitis affecting the neck and chest caused by oak moss present in a partner's aftershave has also been described.[36]

Massage liniment has been reported to cause a contact dermatitis and could therefore potentially cause problems in men, although to date, this has not been described.[37]

Exercise
Exercise induced urticaria and anaphylaxis are well documented.[38] Symptoms may be intermittent and often require an additional factor, such as food sensitivity. Although exercise induced hypersensitivity secondary to sexual intercourse has not been reported to date, the theoretical possibility remains.

Butyl nitrate
The use of inhaled nitrites (''poppers'') by men who have sex with men is well recognised and reports of facial dermatitis associated the use of butyl nitrite have been reported.[39]

Newsprint
The importance of taking a full medical history is highlighted by the report of three women with persistent pruritis vulvae as a result of newspaper printers' ink sensitivity.[40] Their sexual partners were in the habit of reading newspapers in bed at night which was ''often followed by sexual relations including manual manipulation of the vulva.''

NON-SEXUALLY RELATED HYPERSENSITIVITY
Topical medications
Medicaments are well recognised causes of contact dermatitis in patients with leg ulcers and otitis externa but possibly less well appreciated as causes of vulval disease. Marren *et al* found that 29% of women with persisting vulval symptoms failing to respond to standard therapy had evidence of contact hypersensitivity as diagnosed by patch testing.[41] Medicaments were more common sensitisers than cosmetics. The most frequent offenders are ethylenediamine (present in Triadcortyl), framycetin, neomycin, clobetasol propionate, and crotamiton (Eurax).[41 42] The possibility of contact dermatitis should be considered in patients experiencing a

worsening of vulval symptoms while using topical steroids. This may be due to the steroid preparation itself,[43–45] the vehicle,[30] or additives such as an aminoglycoside, preservative, or biocide (for example, chlorocresol).[46]

Topical anaesthetics vary in their ability to cause a contact dermatitis and cross sensitisation between preparations is rare. Lignocaine has low allergenic properties[47] and is less likely to sensitise than other related preparations, such as benzocaine.[42 48]

Topical imidazoles are uncommon causes of contact sensitivity. Those most frequently reported are miconazole, econazole, and tioconazole (treatment for onychomycosis) with cross reactivity being common.[48 49] Clotrimazole may occasionally cause problems although preservatives added to these preparations, such as benzyl alcohol or octyldodecamole, should be considered.[41]

Other preparations used topically on the genitals and reported to cause contact dermatitis, albeit rarely, include clindamycin[50] and aciclovir,[51] although in the latter case other cream constituents, such as propylene glycol, were considered to be the most likely sensitisers.

Feminine hygiene sprays

Feminine hygiene sprays consist of a perfume, an emollient and a propellant. Irritant reactions from fluorinated hydrocarbon propellants sprayed too close to the genitals are more common than allergic reactions.[22] Allergic reactions to the perfume component may be more likely to occur if there is existent skin damage—for example, secondary to candidiasis or dermatitis.[22]

Sexual partners may also be affected, as in the case of a man who developed a dermatitis of the penis, scrotum, and lower abdomen following sexual intercourse with his girlfriend. Patch testing showed a positive reaction to balsam of Peru. Further questioning revealed that his girlfriend used a hygiene spray before intercourse and this was found to contain balsam of Peru.[35]

Bubble baths and scented soaps

Prolonged immersion in baths containing perfumes may induce an irritant vulvitis, particularly in children.[22]

Cosmetics

Potential causes of a genital dermatitis include nail polish, particularly if the vulval skin is touched before the polish is dry[22] and perfumed toilet tissue. Lipstick induced balanitis and penile dermatitis has not been reported but remains a theoretical possibility for men sensitive to octylgallate.[52]

Self adhesive pads

Women with excessive vaginal secretions often use self adhesive pads for comfort and hygiene. A fragrance and disinfecting agent are commonly incorporated into the pad and both may produce contact dermatitis. Sterry and Schmoll reported the case of a woman with genital pruritis who had been using self adhesive pads for several months.[53] Patch testing was positive to the layer of the pad which contained the fragrance and the disinfecting agent (CuII-acetyl acetonate and acetyl acetonate). A similar case has also been described of sensitivity to cinnamic alcohol and cinnamic aldehyde present as a perfume in a deodorant sanitary napkin.[54]

Urine

Irrritant ammoniacal dermatitis should be considered in incontinent patients with genital soreness, particularly if there is a pre-existent genital dermatosis which fails to improve or worsens with treatment.[22]

Colophony

Lewis *et al* reported the case of a violinist with pruritis vulvae caused by a sensitivity to colophony, a substance present in rosin which is used to wax the strings of musical instruments.[55]

Candida

Candida is a well recognised allergen. In vitro tests have documented the release of histamine from rat mast cells by candida antigens[56] and bronchial hypersensitivity to aerosols of *Candida albicans* correlates well with type I but not type IV hypersensitivity. Clinically, candida has been reported to induce asthma and ''tea tasters' cough.'' Genital hypersensitivity to candida has been implicated in some cases of vulvovaginal candidiasis (VVC).[57] Anti-candida IgE antibodies are often present in the vaginal secretions of women with recurrent VVC but not in control women.[57] In addition, there have been reports of partially successful treatment of recurrent VVC by hyposensitisation using subcutaneous injection of increasing doses of candida antigen.[57 58] Male genital hypersensitivity to candida was documented by Catterall who described soreness of the glans penis appearing 6–24 hours after intercourse with women with vaginal candidiasis.[59]

DIAGNOSIS

Vulvitis and balanitis are frequently encountered in clinical practice and in the majority of cases an infective cause or a common genital dermatosis will be identified. Where these are absent, the possibility of an irritant dermatitis or hypersensitivity reaction should be considered. This may be suggested by a history of past or present allergies or a family history of atopy. A history of contact with possible allergens should be ascertained. This may require direct questioning about the use of scented sprays or lubricants before sexual intercourse as patients may feel too embarrassed to volunteer this information. The temporal relation between the onset of symptoms and intercourse may provide useful clues. In cases of seminal fluid hypersensivity, the use of condoms will prevent symptoms and thus may be used as a diagnostic test. Sensitivity to both latex and seminal fluid is likely to be a rare occurrence.[60] Some patients with mild allergic rhinitis have negative skin prick tests and radioallergosorbent test (RAST) but produce a local antibody response together with symptoms on nasal provocation. The role of vulval or vaginal provocation with allergen followed by colposcopic examination of the epithelium has not been assessed but may provide a useful means of assessing allergic vulvo-vaginitis.

Patch testing is the appropriate method for assessing contact dermatitis and is considered a valuable investigative tool for patients with protracted vulval symptoms, particularly if there is no response or a worsening of symptoms while topical steroids are being applied. Patch testing on the mucosa is disappointing since mucous membranes react less clearly to allergens than the skin. In addition, patch testing in this area would prove difficult to perform. Testing should be performed with the British Contact Dermatitis Group (BCDG) standard series, a topical steroid series, medicaments, and other products suggested by the history.

In cases of suspected type I hypersensitivity reactions (for example, latex, seminal fluid), a RAST and skin prick test should be performed. Skin prick tests are considered more sensitive than RASTs but the systemic reaction rate is significant.[61] Neither of these tests are appropriate for assessing contact dermatitis.

Performing and interpreting both skin prick tests and patch tests requires special training and should be only be undertaken in close collaboration with clinicians with

appropriate expertise (for example, dermatologists, allergologists).

MANAGEMENT

The treatment of contact dermatitis and the management of steroid sensitivity are beyond the remit of this paper. Once a potential sensitiser has been identified, avoidance is obviously the optimal approach to management. Condoms should be used in cases of seminal fluid hypersensitivity. Although partial benefit has been reported from hyposensitisation injections[17] this therapeutic technique is viewed with caution in the United Kingdom and, with respect to the use of seminal fluid allergens, may pose practical difficulties. The role of genital biopsy is limited although this may provide histological confirmation of dermatitis and may also help to exclude other pathologies.

CONCLUSION

Genital allergy is uncommon but should be considered as a possible diagnosis in all patients with genital soreness or irritation for which no infection or dermatosis can be identified and in whom symptoms remain unchanged or worsen with treatment. Obtaining an accurate ''allergy history'' may prove difficult and will often require direct questioning regarding possible sensitisers. Type I and IV hypersensitivity reactions are most commonly encountered and can be assessed by performing skin prick testing/RAST or patch testing, respectively. This may require collaboration with an appropriately trained clinician in dermatology or allergology. Once an allergen has been identified, avoidance is the optimal approach to management.

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